Complete Summary

GUIDELINE TITLE

Practice guidelines for the diagnosis and management of skin and soft-tissue infections.

BIBLIOGRAPHIC SOURCE(S)

Stevens DL, Bisno AL, Chambers HF, Everett ED, Dellinger P, Goldstein EJ, Gorbach SL, Hirschmann JV, Kaplan EL, Montoya JG, Wade JC. Practice guidelines for the diagnosis and management of skin and soft-tissue infections. Clin Infect Dis 2005 Nov 15;41(10):1373-406. [236 references] PubMed

GUIDELINE STATUS

This is the current release of the guideline.

COMPLETE SUMMARY CONTENT

SCOPE

DISEASE/CONDITION(S)

Skin and soft-tissue infections including

- Impetigo
- Abscesses, cellulitis, and erysipelas
- Necrotizing skin and soft-tissue infections
- Infections following animal and human bites
- Soft-tissue infections following animal contact
- Surgical site infections
- Infections in the immune-compromised host
- Infections related to iatrogenic procedures

GUIDELINE CATEGORY

Diagnosis
Management
Treatment

CLINICAL SPECIALTY

Dermatology
Family Practice
Infectious Diseases
Internal Medicine
Pediatrics
Surgery

INTENDED USERS

Physicians

GUIDELINE OBJECTIVE(S)

To provide recommendations for diagnosis and management of skin and soft-tissue infections in otherwise healthy hosts and compromised hosts of all age groups

TARGET POPULATION

Patients of all ages with skin and soft-tissue infections

INTERVENTIONS AND PRACTICES CONSIDERED

Diagnostic Assessment

1. History and physical examination
2. Laboratory tests (blood culture and drug susceptibility tests, complete blood cell count with differential, creatinine, bicarbonate, creatine phosphokinase, and C-reactive protein levels)
3. Gram stain and culture of needle aspiration/punch biopsy specimens
4. Immunohistochemistry and polymerase chain reactions (PCR) of specimens
5. Surgical consultation for inspection, exploration and/or drainage

Treatment

1. Antimicrobial selection and administration
2. Antimicrobial agents
3. Adjunct therapy
    - Granulocyte colony stimulating factor/granulocyte macrophage colony stimulating factor (G-CSF/GM-CSF)
    - Granulocyte therapy
4. Surgical intervention (drainage, debridement)
5. Treatment for immunocompromised hosts

MAJOR OUTCOMES CONSIDERED

- Morbidity and mortality
- Healing time
- Response to treatment
- Treatment time
- Hospitalization rate and duration of hospital stay
- Incidence of relapse or recurrence

METHODOLOGY

METHODS USED TO COLLECT/SELECT EVIDENCE

Searches of Electronic Databases

DESCRIPTION OF METHODS USED TO COLLECT/SELECT THE EVIDENCE

Not stated

NUMBER OF SOURCE DOCUMENTS

Not stated

METHODS USED TO ASSESS THE QUALITY AND STRENGTH OF THE EVIDENCE

Weighting According to a Rating Scheme (Scheme Given)

RATING SCHEME FOR THE STRENGTH OF THE EVIDENCE

Quality of Evidence

I. Evidence from ≥1 properly randomized, controlled trial
II. Evidence from ≥1 well-designed clinical trial, without randomization; from cohort or case-controlled analytic studies (preferably from >1 center); from multiple time-series; or from dramatic results from uncontrolled experiments
III. Evidence from opinions of respected authorities, based on clinical experience, descriptive studies, or reports of expert committees

METHODS USED TO ANALYZE THE EVIDENCE

Review

DESCRIPTION OF THE METHODS USED TO ANALYZE THE EVIDENCE

Not stated

METHODS USED TO FORMULATE THE RECOMMENDATIONS

Expert Consensus

DESCRIPTION OF METHODS USED TO FORMULATE THE RECOMMENDATIONS

Not stated

RATING SCHEME FOR THE STRENGTH OF THE RECOMMENDATIONS

Strength of Recommendation

A. Good evidence to support a recommendation for use; should always be offered
B. Moderate evidence to support a recommendation for use; should generally be offered
C. Poor evidence to support a recommendation; optional
D. Moderate evidence to support a recommendation against use; should generally not be offered
E. Good evidence to support a recommendation against use; should never be offered

COST ANALYSIS

A formal cost analysis was not performed and published cost analyses were not reviewed.

METHOD OF GUIDELINE VALIDATION

Peer Review

DESCRIPTION OF METHOD OF GUIDELINE VALIDATION

Not stated

RECOMMENDATIONS

MAJOR RECOMMENDATIONS

Note from the National Guideline Clearinghouse (NGC): The following comes from the Executive Summary of the guideline. Please see the full guideline for additional details about the topics discussed below.

The strength of recommendation (A-E) and quality of evidence (I-III) are defined at the end of the "Major Recommendations" field.

Executive Summary

Soft-tissue infections are common, generally of mild to modest severity, and are easily treated with a variety of agents. An etiologic diagnosis of simple cellulitis is frequently difficult and generally unnecessary for patients with mild signs and

symptoms of illness. Clinical assessment of the severity of infection is crucial, and several classification schemes and algorithms have been proposed to guide the clinician. However, most clinical assessments have been developed from either retrospective studies or from an author's own "clinical experience," illustrating the need for prospective studies with defined measurements of severity coupled to management issues and outcomes.

Until then, it is the recommendation of this committee that patients with soft-tissue infection accompanied by signs and symptoms of systemic toxicity (e.g., fever or hypothermia, tachycardia [heart rate >100 beats/min], and hypotension [systolic blood pressure, <90 mm Hg or 20 mm Hg below baseline]) have blood drawn to determine the following laboratory parameters: results of blood culture and drug susceptibility tests, complete blood cell count with differential, and creatinine, bicarbonate, creatine phosphokinase, and C-reactive protein levels. In patients with hypotension and/or an elevated creatinine level, low serum bicarbonate level, elevated creatine phosphokinase level (2-3 times the upper limit of normal), marked left shift, or a C-reactive protein level >13 mg/L, hospitalization should be considered and a definitive etiologic diagnosis pursued aggressively by means of procedures such as Gram stain and culture of needle aspiration or punch biopsy specimens, as well as requests for a surgical consultation for inspection, exploration, and/or drainage. Other clues to potentially severe deep soft-tissue infection include the following: (1) pain disproportionate to the physical findings, (2) violaceous bullae, (3) cutaneous hemorrhage, (4) skin sloughing, (5) skin anesthesia, (6) rapid progression, and (7) gas in the tissue. Unfortunately, these signs and symptoms often appear later in the course of necrotizing infections. In these cases, emergent surgical evaluation is of paramount importance for both diagnostic and therapeutic reasons.

Emerging antibiotic resistance among Staphylococcus aureus (methicillin resistance) and Streptococcus pyogenes (erythromycin resistance) are problematic, because both of these organisms are common causes of a variety of skin and soft-tissue infections and because empirical choices of antimicrobials must include agents with activity against resistant strains. Minor skin and soft-tissue infections may be empirically treated with semisynthetic penicillin, first-generation or second-generation oral cephalosporins, macrolides, or clindamycin (A-I); however, 50% of methicillin-resistant S. aureus (MRSA) strains have inducible or constitutive clindamycin resistance. Most community-acquired MRSA strains remain susceptible to trimethoprim-sulfamethoxazole and tetracycline, though treatment failure rates of 21% have been reported in some series with doxycycline or minocycline. Therefore, if patients are sent home receiving these regimens, it is prudent to reevaluate them in 24-48 hours to verify a clinical response. Progression despite receipt of antibiotics could be due to infection with resistant microbes or because a deeper, more serious infection exists than was previously realized.

Patients who present to the hospital with severe infection or whose infection is progressing despite empirical antibiotic therapy should be treated more aggressively, and the treatment strategy should be based upon results of appropriate Gram stain, culture, and drug susceptibility analysis. In the case of S. aureus, the clinician should assume that the organism is resistant, because of the high prevalence of community-associated MRSA strains, and agents effective

against MRSA (i.e., vancomycin, linezolid, or daptomycin) should be used (A-I). Stepdown to treatment with other agents, such as tetracycline or trimethoprim-sulfamethoxazole, for MRSA infection may be possible, based on results of susceptibility tests and after an initial clinical response. In the United States, not all laboratories perform susceptibility testing on S. pyogenes. However, the Centers for Disease Control and Prevention has provided national surveillance data that suggest a gradual trend of increasing macrolide resistance of S. pyogenes from 4%-5% in 1996-1998 to 8%-9% in 1999-2001. Of interest, 99.5% of strains remain susceptible to clindamycin, and 100% are susceptible to penicillin.

Impetigo, Erysipelas, and Cellulitis

Impetigo may be caused by infection with S. aureus and/or S. pyogenes. The decision of how to treat impetigo depends on the number of lesions, their location (face, eyelid, or mouth), and the need to limit spread of infection to others. The best topical agent is mupirocin (A-I), although resistance has been described; other agents, such as bacitracin and neomycin, are considerably less effective treatments. Patients who have numerous lesions or who are not responding to topical agents should receive oral antimicrobials effective against both S. aureus and S. pyogenes (A-I) (see the table below entitled "Antimicrobial Therapy for Impetigo and for Skin and Soft-Tissue Infections"). Although rare in developed countries (<1 case/1,000,000 population per year), glomerulonephritis following streptococcal infection may be a complication of impetigo caused by certain strains of S. pyogenes, but no data demonstrate that treatment of impetigo prevents this sequela.

Classically, erysipelas is a fiery red, tender, painful plaque with well-demarcated edges and is commonly caused by streptococcal species, usually S. pyogenes.

Cellulitis may be caused by numerous organisms that are indigenous to the skin or to particular environmental niches. Cellulitis associated with furuncles, carbuncles, or abscesses is usually caused by S. aureus. In contrast, cellulitis that is diffuse or unassociated with a defined portal is most commonly caused by streptococcal species. Important clinical clues to other causes include physical activities, trauma, water contact, and animal, insect, or human bites. In these circumstances appropriate culture material should be obtained, as they should be in patients who do not respond to initial empirical therapy directed against S. aureus and S. pyogenes and in immunocompromised hosts. Unfortunately, aspiration of skin is not helpful in 75%-80% of cases of cellulitis, and results of blood cultures are rarely positive (<5% of cases).

Penicillin, given either parenterally or orally depending on clinical severity, is the treatment of choice for erysipelas (A-I). For cellulitis, a penicillinase-resistant semisynthetic penicillin or a first-generation cephalosporin should be selected (A-I), unless streptococci or staphylococci resistant to these agents are common in the community. For penicillin-allergic patients, choices include clindamycin or vancomycin.

Lack of clinical response could be due to unusual organisms, resistant strains of staphylococcus or streptococcus, or deeper processes, such as necrotizing fasciitis or myonecrosis. In patients who become increasingly ill or experience increasing

toxicity, necrotizing fasciitis, myonecrosis, or toxic shock syndrome should be considered, an aggressive evaluation initiated, and antibiotic treatment modified, on the basis of Gram stain results, culture results, and antimicrobial susceptibilities of organisms obtained from surgical specimens.

Antimicrobial Therapy for Impetigo and for Skin and Soft-Tissue Infections

Antibiotic therapy, by disease	Comment
Impetigo	
Dicloxacillin	
Cephalexin	
Erythromycin	Some strains of Staphylococcus aureus and Streptococcus pyogenes may be resistant
Clindamycin	
Amoxicillin/clavulanate	
Mupirocin ointment	For patients with a limited number of lesions
MSSA SSTI	
Nafcillin or oxacillin	Parental drug of choice; inactive against MRSA
Cefazolin	For penicillin-allergic patients, except those with immediate hypersensitivity reactions
Clindamycin	Bacteriostatic; potential of cross-resistance and emergence of resistance in erythromycin-resistant strains; inducible resistance in MRSA
Dicloxacillin	Oral agent of choice for methicillin-susceptible strains
Cephalexin	For penicillin-allergic patients, except those with immediate hypersensitivity reactions
Doxycycline, minocycline	Bacteriostatic; limited recent clinical experience
TMP-SMZ	Bactericidal; efficacy poorly documented
MRSA SSTI	
Vancomycin	For penicillin-allergic patients; parenteral drug of choice for treatment of infections caused by MRSA
Linezolid	Bacteriostatic; limited clinical experience; no cross-resistance with other antibiotic classes; expensive; may eventually replace other second-line agents as a preferred agent for oral therapy of MRSA infections
Clindamycin	Bacteriostatic; potential of cross-resistance and emergence of resistance in erythromycin- resistant strains; inducible resistance in MRSA
Daptomycin	Bactericidal; possible myopathy
Doxycycline, minocycline	Bacteriostatic, limited recent clinical experience
TMP-SMZ	Bactericidal; limited published efficacy data

Note: MRSA, methicillin-resistant S. aureus; MSSA, methicillin-susceptible S. aureus; SSTI, skin and soft-tissue infection; TMP-SMZ, trimethoprim-sulfamethoxazole.

Necrotizing Infections

Necrotizing fasciitis may be monomicrobial and caused by S. pyogenes, Vibrio vulnificus, or Aeromonas hydrophila. Recently, necrotizing fasciitis was described in a patient with MRSA infection. Polymicrobial necrotizing fasciitis may occur following surgery or in patients with peripheral vascular disease, diabetes mellitus, decubitus ulcers, and spontaneous mucosal tears of the gastrointestinal or gastrourinary tract (i.e., Fournier gangrene). As with clostridial myonecrosis, gas in the deep tissues is frequently found in these mixed infections.

Gas gangrene is a rapidly progressive infection caused by Clostridium perfringens, Clostridium septicum, Clostridium histolyticum, or Clostridium novyi. Severe penetrating trauma or crush injuries associated with interruption of the blood supply are the usual predisposing factors. C. perfringens and C. novyi infections have recently been described among heroin abusers following intracutaneous injection of black tar heroin. C. septicum, a more aerotolerant Clostridium species, may cause spontaneous gas gangrene in patients with colonic lesions (such as those due to diverticular disease), adenocarcinoma, or neutropenia.

Necrotizing fasciitis and gas gangrene may cause necrosis of skin, subcutaneous tissue, and muscle. Cutaneous findings of purple bullae, sloughing of skin, marked edema, and systemic toxicity mandate prompt surgical intervention. For severe group A streptococcal and clostridial necrotizing infections, parenteral clindamycin and penicillin treatment is recommended (A-II). A variety of antimicrobials directed against aerobic gram-positive and gram-negative bacteria, as well as against anaerobes, may be used in mixed necrotizing infections (B-II).

Infections Following Animal or Human Bites

Animal bites account for 1% of all emergency department visits, and dog bites are responsible for 80% of such cases. Although Pasteurella species are the most common isolates, cat and dog bites contain an average of 5 different aerobic and anaerobic bacteria per wound, often including S. aureus, Bacteroides tectum, and Fusobacterium, Capnocytophaga, and Porphyromonas species. The decision to administer oral or parenteral antibiotics depends on the depth and severity of the wound and on the time since the bite occurred. Patients not allergic to penicillin should receive treatment with oral amoxicillin-clavulanate or with intravenous ampicillin-sulbactam or ertapenem (B-II), because agents such as dicloxacillin, cephalexin, erythromycin, and clindamycin have poor activity against Pasteurella multocida. Although cefuroxime, cefotaxime, and ceftriaxone are effective against P. multocida, they do not have good anaerobic spectra. Thus, cefoxitin or carbapenem antibiotics could be used parenterally in patients with mild penicillin allergies. Patients with previous severe reactions can receive oral or intravenous doxycycline, trimethoprim-sulfamethoxazole, or a fluoroquinolone plus clindamycin.

Human bites may occur from accidental injuries, purposeful biting, or closed fist injuries. The bacteriologic characteristics of these wounds are complex but include infection with aerobic bacteria, such as streptococci, S. aureus, and Eikenella corrodens, as well as with multiple anaerobic organisms, including Fusobacterium, Peptostreptococcus, Prevotella, and Porphyromonas species. E. corrodens is resistant to first-generation cephalosporins, macrolides, clindamycin, and aminoglycosides. Thus, intravenous treatment with ampicillin-sulbactam or cefoxitin is the best choice (B-III).

Infections Associated with Animal Contact

Infections associated with animal contact, although uncommon, are frequently severe, sometimes lethal, and diagnostically challenging. The potential use of Bacillus anthracis, Francisella tularensis, and Yersinia pestis for bioterrorism has generated great interest in rapid diagnostic techniques, because early recognition and treatment are essential. Doxycycline or ciprofloxacin therapy is recommended in standard doses for nonpregnant adults and children 18 years of age, pending identification of the offending agent (B-III).

Adults and children who receive a diagnosis of tularemia should receive an aminoglycoside, preferably streptomycin or gentamicin, for 7-10 days. In mild cases, doxycycline or tetracycline for 14 days is recommended (B-III) (comments regarding treatment of children <8 years of age are specified in table 3 of the original guideline document). Patients with bubonic plague should receive streptomycin, tetracycline, or chloramphenicol for 10-14 days and should be placed in isolation for 48 hours after initiation of treatment, because some patients may develop secondary pneumonic plague (B-III).

Data regarding antibiotic efficacy for treatment of cat-scratch disease are inconclusive, although 1 small study demonstrated more-rapid lymph node regression in patients receiving azithromycin, compared with patients receiving no treatment. Cutaneous bacillary angiomatosis has not been systematically studied, but treatment with erythromycin or doxycycline in standard doses for 4 weeks has been effective in very small series (B-III).

On the basis of very incomplete data, erysipeloid is best treated with oral penicillin or amoxicillin for 10 days (B-III). E. rhusiopathiae is resistant in vitro to vancomycin, teicoplanin, and daptomycin (E-III).

Surgical Site Infections

Surgical soft-tissue infections include those occurring postoperatively and those severe enough to require surgical intervention for diagnosis and treatment. The algorithm presented in the original guideline document clearly indicates that surgical site infection rarely occurs during the first 48 hours after surgery, and fever during that period usually arises from noninfectious or unknown causes. In contrast, after 48 hours, surgical site infection is a more common source of fever, and careful inspection of the wound is indicated. For patients with a temperature <38.5 degrees C and without tachycardia, observation, dressing changes, or opening the incision site suffices. Patients with a temperature >38.5 degrees C or a heart rate >110 beats/minute generally require antibiotics as well as opening of the suture line. Infections developing after surgical procedures involving nonsterile tissue, such as colonic, vaginal, biliary, or respiratory mucosa, may be caused by a combination of aerobic and anaerobic bacteria. These infections can rapidly progress and involve deeper structures than just the skin, such as fascia, fat, or muscle (see table below entitled "Antibiotic Choices for Incisional Surgical Site Infections").

Antibiotic Choices for Incisional Surgical Site Infections (SSIs)

Antibiotic Therapy for SSIs, By Site of Operation

Intestinal or genital tract
 	
• Single agents
  • Cefoxitin
  • Ceftizoxime
  • Ampicillin/sulbactam
  • Ticarcillin/clavulanate
  • Piperacillin/tazobactam
  • Imipenem/cilastatin
  • Meropenem
  • Ertapenem
• Combination agents
  • Facultative and aerobic activity
  • Fluoroquinolone
  • Third-generation cephalosporin
  • Aztreonam[a]
  • Aminoglycoside
  • Anaerobic activity
  • Clindamycin
  • Metronidazole[a]
  • Chloramphenicol
  • Penicillin agent plus beta-lactamase inhibitor
 	
Nonintestinal
 	
• Trunk and extremities away from axilla or perineum
  • Oxacillin
  • First-generation cephalosporin
• Axillary or perineum
  • Cefoxitin
  • Ampicillin/sulbactam
  • Other single agents as described above for intestinal and genital operations

[a] Do not combine aztreonam with metronidazole, because this combination has no activity against gram-positive cocci.

Infections in the Immunocompromised Host

Skin and soft tissues are common sites of infection in compromised hosts and usually pose major diagnostic challenges for the following 3 reasons: (1) infections are caused by diverse organisms, including organisms not ordinarily considered to be pathogens in otherwise healthy hosts; (2) infection of the soft tissues may occur as part of a broader systemic infection; and (3) the degree and type of immune deficiency attenuate the clinical findings. The importance of establishing a diagnosis and performing susceptibility testing is crucial, because many infections are hospital acquired, and mounting resistance among both gram-positive and gram-negative bacteria makes dogmatic empirical treatment regimens difficult, if not dangerous. In addition, fungal infections may present with cutaneous findings.

Immunocompromised patients who are very ill or experiencing toxicity typically require very broad-spectrum empirical agents that include specific coverage for resistant gram-positive bacteria, such as MRSA (e.g., vancomycin, linezolid, daptomycin, or quinupristin/dalfopristin). Coverage for gram-negative bacteria may include monotherapy with a cephalosporin possessing activity against Pseudomonas species, with carbapenems, or with a combination of either a fluoroquinolone or an aminoglycoside plus either an extended-spectrum penicillin or cephalosporin.

Infections in patients with cell-mediated immunodeficiency (such as that due to Hodgkin disease, lymphoma, human immunodeficiency virus [HIV] infection, bone marrow transplantation, and receipt of long-term high-dose immunosuppressive therapy) can be caused by either common or unusual bacteria, viruses, protozoa, helminths, or fungi. Although infection may begin in the skin, cutaneous lesions can also be the result of hematogenous seeding. A well planned strategy for prompt diagnosis, including biopsy and aggressive treatment protocols, is essential. Diagnostic strategies require laboratory support capable of rapid processing and early detection of bacteria (including Mycobacteria and Nocardia species), viruses, and fungi. The algorithm presented in the original guideline document provides an approach to diagnosis and treatment. The empirical antibiotic guidelines are based on results of clinical trials, national surveillance antibiograms, and consensus meetings. Because antimicrobial susceptibilities vary considerably across the nation, clinicians must base empirical treatment on the antibiograms in their own location.

Microbiologic cultures are important in establishing a specific diagnosis, and testing the drug susceptibility of organisms is critical for optimal antimicrobial treatment. This guideline offers recommendations for empirical treatment of specific community-acquired and hospital-acquired infections. Nonetheless, therapy may fail for several reasons: (1) the initial diagnosis and/or treatment chosen is incorrect, (2) the etiologic agent from a given locale is resistant to antibiotics, (3) antimicrobial resistance develops during treatment, and (4) the infection is deeper and more complex than originally estimated.

Skin and Soft-tissue Infections in the Immune Compromised Host: Treatment and Management

Predisposing factor, pathogen	Type of therapy	Duration of therapy	Frequency or reason for surgery	Adjunct
Neutropenia				
Initial infection				
Bacteria:				
Gram negative	Monotherapy or antibiotic combination	7-14 days	Rare	G-CSF/GM-CSF; granulocyte therapy[a]
Gram positive	Pathogen specific	7-10 days	Rare	No
Subsequent infection				
Antibiotic-resistant	Pathogen specific	7-14 days	Rare	G-CSF/GM-

Predisposing factor, pathogen	Type of therapy	Duration of therapy	Frequency or reason for surgery	Adjunct
bacteria				CSF;[b] granulocyte therapy[a]
Fungi	Amphotericin B, voriconazole, or caspofungin	Clinical and radiologic resolution	For localized infection	Catheter removal; G-CSF/GM-CSF;[b] granulocyte therapy[a]
	Cellular immune deficiency			
Bacteria				
Nocardia species	Trimethoprim-sulfamethoxazole or sulfadiazine	3-12 months	Rare	No
Atypical mycobacteria	Antibiotic combination (including a macrolide)	3-6 weeks	Yes	No
Fungi				
Cryptococcus species	Amphotericin B plus 5-fluorocytosine or fluconazole	8-12 weeks	No	No
Histoplasma species	Amphotericin B or itraconazole			
Viruses				
Varicella-zoster virus	Acyclovir famciclovir valacyclovir	7-10 days	No	No
Herpes simplex virus	Acyclovir famciclovir valacyclovir	7 days	No	No
Cytomegalovirus	Ganciclovir	21 days	No	No

Note: G-CSF, granulocyte colony-stimulating factor; GM-CSF, granulocyte-monocyte colony-stimulating factor.

[a]Use if gram-negative bacillary infection is unresponsive to appropriate antimicrobial therapy or if the patient has invasive fungal infection.

[b]Progressive infection, pneumonia, and invasive fungal infection.

Definitions:

Quality of Evidence

I. Evidence from ≥1 properly randomized, controlled trial
II. Evidence from ≥1 well-designed clinical trial, without randomization; from cohort or case-controlled analytic studies (preferably from >1 center); from multiple time-series; or from dramatic results from uncontrolled experiments

III. Evidence from opinions of respected authorities, based on clinical experience, descriptive studies, or reports of expert committees

Strength of Recommendation

A. Good evidence to support a recommendation for use; should always be offered
B. Moderate evidence to support a recommendation for use; should generally be offered
C. Poor evidence to support a recommendation; optional
D. Moderate evidence to support a recommendation against use; should generally not be offered
E. Good evidence to support a recommendation against use; should never be offered

CLINICAL ALGORITHM(S)

A clinical algorithm for the management and treatment of surgical site infections is provided in the original guideline document.

EVIDENCE SUPPORTING THE RECOMMENDATIONS

TYPE OF EVIDENCE SUPPORTING THE RECOMMENDATIONS

The type of supporting evidence is identified and graded for most recommendations (see "Major Recommendations").

BENEFITS/HARMS OF IMPLEMENTING THE GUIDELINE RECOMMENDATIONS

POTENTIAL BENEFITS

Appropriate and successful treatment of skin and soft tissue infections, resulting in the resolution of signs and symptoms, prevention of complications

POTENTIAL HARMS

Side effects of pharmacological agents

CONTRAINDICATIONS

CONTRAINDICATIONS

- The use of fluoroquinolones is contraindicated by the US Food and Drug Administration for children and adolescents <18 years of age. It should also be noted that tetracyclines are rarely used in children <8 years of age. Alternatives should be strongly considered for these 2 antibiotics.
- Penicillin-allergic pregnant women constitute a special population, because tetracyclines, sulfa compounds (during late pregnancy), and metronidazole are contraindicated.

IMPLEMENTATION OF THE GUIDELINE

DESCRIPTION OF IMPLEMENTATION STRATEGY

An implementation strategy was not provided.

IMPLEMENTATION TOOLS

Clinical Algorithm

For information about availability, see the "Availability of Companion Documents" and "Patient Resources" fields below.

INSTITUTE OF MEDICINE (IOM) NATIONAL HEALTHCARE QUALITY REPORT CATEGORIES

IOM CARE NEED

Getting Better

IOM DOMAIN

Effectiveness

IDENTIFYING INFORMATION AND AVAILABILITY

BIBLIOGRAPHIC SOURCE(S)

Stevens DL, Bisno AL, Chambers HF, Everett ED, Dellinger P, Goldstein EJ, Gorbach SL, Hirschmann JV, Kaplan EL, Montoya JG, Wade JC. Practice guidelines for the diagnosis and management of skin and soft-tissue infections. Clin Infect Dis 2005 Nov 15;41(10):1373-406. [236 references] PubMed

ADAPTATION

Not applicable: The guideline was not adapted from another source.

DATE RELEASED

2005 Nov 15

GUIDELINE DEVELOPER(S)

Infectious Diseases Society of America - Medical Specialty Society

SOURCE(S) OF FUNDING

Infectious Diseases Society of America (IDSA)

GUIDELINE COMMITTEE

Infectious Diseases Society of America (IDSA) Standards and Practice Guidelines Committee

COMPOSITION OF GROUP THAT AUTHORED THE GUIDELINE

Authors: Dennis L. Stevens, Infectious Diseases Section, Veterans Affairs Medical Center, Boise, Idaho, University of Washington School of Medicine, Seattle; Alan L. Bisno, University of Miami Miller School of Medicine, Miami, Florida; Henry F. Chambers, Infectious Diseases, San Francisco General Hospital, University of California-San Francisco, San Francisco; E. Dale Everett, University of Missouri Health Science Center, University of Missouri, Columbia; Patchen Dellinger, Department of Surgery; Ellie J. C. Goldstein, R. M. Alden Research Laboratory, Santa Monica, University of California, Los Angeles School of Medicine, Los Angeles; Sherwood L. Gorbach, Tufts University School of Medicine, Boston, Massachusetts; Jan V. Hirschmann, University of Washington School of Medicine, Seattle Veterans Affairs Medical Center, Seattle, Washington; Edward L. Kaplan, University of Minnesota Medical School, Division of Epidemiology, University of Minnesota School of Public Health, Minneapolis, Minnesota; Jose G. Montoya, Department of Medicine, Division of Infectious Diseases and Geographic Medicine, Stanford University School of Medicine, Research Institute, Palo Alto Medical Foundation, Palo Alto, California; James C. Wade, Division of Neoplastic Diseases and Related Disorders, Medical College of Wisconsin, Milwaukee, Wisconsin

FINANCIAL DISCLOSURES/CONFLICTS OF INTEREST

D.L.S. has received research funding from Wyeth, Lederle, Pfizer, Amgen, Roche, and Cubist and has served as a consultant for Schering Plough, Pfizer, and Arpida. A.L.B. has served as a consultant for Merck, Cubist, Pharmacia, and Schering Plough. H.F.C. has received grant or research support from Ortho-McNeil and Cubist, has served as a consultant for or on the advisory board of Ortho-McNeil and Osmotics, and has received honoraria from Basilea. P.D. has received grants for clinical research from, served on the advisory board of, and/or lectured for honoraria from GlaxoSmithKline, Bayer, Eli Lily, Merck, Wyeth-Ayerst, Bristol-Myers Squibb, AstraZeneca, Pfizer, Aventis, HoffmanLa Roche, Arrow, Ortho-McNeil, Parke-Davis, Abbot, ICOS, Immunex, Chiron, Searle, Cubist, Virucon, InterMune, Peninsula, Johnson & Johnson, and BRAHMS. E.J.C.G. has served as a consultant for, is on the speakers' bureaus of, and/or has received research support from Merck, Aventis, Cubist, Bayer, Schering Plough, GlaxoSmithKline, Ortho-McNeil, and Vicuron and has served on the scientific advisory board of Merck, Bayer, and Schering Plough. J.G.M. has served on the speakers' bureaus of Merck, Pfizer, Enzon, Aventis, and Schering Plough. All other authors: no conflicts.

GUIDELINE STATUS

This is the current release of the guideline.

GUIDELINE AVAILABILITY

Electronic copies: Available from the Infectious Disease Society of America (IDSA) Web site:

- HTML Format
- Portable Document Format (PDF)
- Post Script (PS)

Print copies: Available from Infectious Diseases Society of America, 66 Canal Center Plaza, Suite 600, Alexandria, VA 22314.

AVAILABILITY OF COMPANION DOCUMENTS

The following is available:

- Kish MA. Guide to development of practice guidelines. Clin Infect Dis 2001 Mar 15;32(6):851-4.

Electronic copies: Available from the Infectious Diseases Society of America (IDSA) Web site.

Print copies: Available from Infectious Diseases Society of America, 66 Canal Center Plaza, Suite 600, Alexandria, VA 22314.

PATIENT RESOURCES

None available

NGC STATUS

This summary was completed by ECRI on December 7, 2005. The information was verified by the guideline developer on January 24, 2006.

COPYRIGHT STATEMENT

DISCLAIMER

NGC DISCLAIMER

The National Guideline Clearinghouse™ (NGC) does not develop, produce, approve, or endorse the guidelines represented on this site.

All guidelines summarized by NGC and hosted on our site are produced under the auspices of medical specialty societies, relevant professional associations, public or private organizations, other government agencies, health care organizations or plans, and similar entities.

Guidelines represented on the NGC Web site are submitted by guideline developers, and are screened solely to determine that they meet the NGC Inclusion Criteria which may be found at http://www.guideline.gov/about/inclusion.aspx.

NGC, AHRQ, and its contractor ECRI make no warranties concerning the content or clinical efficacy or effectiveness of the clinical practice guidelines and related materials represented on this site. Moreover, the views and opinions of developers or authors of guidelines represented on this site do not necessarily state or reflect those of NGC, AHRQ, or its contractor ECRI, and inclusion or hosting of guidelines in NGC may not be used for advertising or commercial endorsement purposes.

Readers with questions regarding guideline content are directed to contact the guideline developer.

Date Modified: 10/9/2006